vomiting, headache, or optic neuritis. The diagnosis was a lesion, probably a neoplasm, in the ascending frontal convolution. The patient later developed general convulsions and a full hemiplegia of the left side.

He died after an operation. A gliosis was found which invaded much of the frontal lobe and backwards slightly beyond the ascending parietal convolution.

The features of the case were: the presence of paresthesia or subjective sensory phenomena for many weeks before objective signs of sensory disturbance appeared in the same areas; the diagnosis of a probable tumor from the presence of an advancing paralysis with symptoms of corresponding cortical irritation, although all the general symptoms of tumor were absent; the presence of data from which an extensive growth of the glosis in seven to eight weeks could be estimated.

A CASE OF APRAXIA WITH AUTOPSY.

By John H. W. Rhein, M.D.

(To be published in this journal.)

Man of fifty-five developed progressive loss of eyesight, together with inability to recognize the position of his body and limbs; loss of the sense of location generally; loss of tactile sense in the left hand, and disturbance of the temperature sense in the left hand. The stereognostic perception was impaired on both sides. Finally, there was a contracture of the left arm and hand, with rigidity of the left leg, some rigidity of the right arm and leg, but not to the point of contracture. Progressive mental failure.

Autopsy revealed an area of softening in the cortex, in the occipito-parietal region on the right side. Sections of the brain showed degeneration in the white matter of both occipital lobes, and part of the parietal lobes.

Discussion of apraxia.

Discussion.

Dr. Joseph Collins said that the case reported had very decided interest for him because the patient had been for a brief time under his observation and because he had utilized him both at the American Neurological Association meeting in Boston two years ago and at the meeting of the New York and Philadelphia Neurological Societies in this city a year or so ago, as an illustration of a clinical type of cerebral arteriosclerosis which he has frequently encountered and to which he had desired to call attention. This patient was considered by some physicians who had observed him to have a brain lesion of the nature of a neoplasm. When Dr. Collins saw him he was receiving antisyphilitic medication. Dr. Collins' diagnosis was made in regard to his lesion, entirely aside from his agnosia, apraxia, and loss of the sense of position which conditions he had then much as Dr. Rhein related them to-day. Dr. Collins maintained then and later that the patient conformed in practically all the clinical manifestations with a type of arteriosclerosis which he had described at the meetings mentioned and he thinks that the autopsy corroborates that view. So far as Dr. Collins could judge from the brief report which was given by Dr. Rhein the morbid condition was in reality a softening, probably dependent upon a widespread arteriosclerosis, although he did not recall that anything had been said about the evidences to be found on further microscopical examination of the brain. The softening which Dr. Rhein describes is probably an encephalomalacia dependent upon an arteriosclerosis. Dr. Collins called attention a year ago to the patient's face and features, his attitude of fixation, an apparent transformation from an animate being to an inanimate object, a fixation of face and feature that reminded of paralysis agitans and he pointed out at that time that the complex of symptoms could be explained only by positing such an extensive lesion as arteriosclerosis. The focal symptoms which Dr. Rhein had discussed were obviously dependent upon the softening which he described, but the general symptoms he believed could be interpreted only on the supposition that there was some widespread disease such as arteriosclerosis. Surely many of the symptoms which the patient had had could not be ascribed to the softening. Dr. Collins asked whether the autopsy showed arterial lesions, whether serial sections had been made of the brain ganglia and how much degeneration existed.

Dr. A. Gordon said that in cases of hemiplegia of cortical or subcortical origin, it is frequently noticed that there is disturbance of function of the opposite side. He asked whether a systematic histological study of the corpus callosum was made by Dr. Rhein, as it appears that symptoms were present on both sides. Apraxia is not infrequently the result of involvement of the white matter, particularly of the corpus callosum.

In closing the discussion, Dr. Rhein, in answer to Dr. Collins' query, stated that there was general arteriosclerosis present. Microscopic studies had been carefully made. The arteriosclerosis was not very intense, consisting of moderate perivascular distension and infiltration. This was especially noted in the necrotic area. It was also present in the degenerated white matter.

In further answer to Dr. Collins' remarks, Dr. Rhein stated that, in his opinion, the chief importance of this arteriosclerosis was in its localization, rather than the lesion itself. The visual disturbances, the absence of degeneration in the calcarine region, the degeneration of the white matter of the occipital lobe, with partial degeneration of the inferior longitudinal bundle and optic radiations, present a picture which is not in accord with the present views of the pathology of cortical blindness. He had no explanation for this, except that perhaps the calcarine cortex did not govern primary vision independently, but that it operated in conjunction with other portions of the occipital lobe.

Dr. Gordon's point was taken up in detail in Dr. Rhein's paper. A study of the corpus callosum was made, and this phase of the subject was considered in discussion of the cases of Liepmann, Van Vleuten and Mass.

TREATMENT OF FACIAL SPASM BY MEANS OF INJECTIONS OF ALCOHOL. REPORT OF THREE CASES

By Hugh T. Patrick, M.D.

Brief report of three cases of typical facial spasm treated by injections of alcohol into the facial nerve at the stylo-mastoid foramen. Good results in two, failure to reach the nerve in the third. Exhibition of photographs.

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DISCUSSION.

Dr. J. J. Putnam asked whether Dr. Patrick considers that the injection of the alcohol induces a special pathological condition of the nerve and how he explains the result as compared, for example, with the failures which characterized his own efforts some years ago to treat facial spasm by nerve stretching.

Dr. Gordon asked whether Dr. Patrick had any experience with his treatment in hemi-spasms following peripheral facial palsy.

Dr. H. M. Thomas said that he had seen a number of cases in which operation was performed for facial spasm, and he thinks the facial palsy is as bad as the original condition. The only case of spasm in which a cure had been effected was one in which Dr. Cushing made an anastomosis of the facial and the spinal accessory nerves. That patient is practically well, although she has not recovered complete voluntary power. Dr. Thomas said the emotional power came back perfectly and came back before the voluntary power in the paralyzed face.

Dr. G. L. Walton emphasized the presence of other obsessive tendencies in the victims of tic. Sufferers from spasm may be neurotic but not necessarily obsessive. He spoke of the importance of carefully correcting refractive error before resorting to direct attack upon the seventh nerve. He mentioned an obstinate case of many years standing completely cured by a final adjustment of glasses and muscular exercises for insufficiency.

Dr. Charles E. Beevor said he was glad Dr. Patrick made the distinction between tic and facial spasm, the spasm is anatomical movement of muscles, whereas tic is functional. An important point in regard to the treatment is in regard to hitting the nerve. He supposed the evidence was that if the nerve is not entered one cannot get any paralysis. Is there any twitching the moment the needle goes into the nerve? The motor nerve if you irritate it will twitch. Dr. Beevor thinks that we ought to get twitching of the nerve the moment the needle enters it. He wished to know how much alcohol Dr. Patrick used, whether one or two injections, and whether he employed absolute alcohol.

Dr. J. Ramsay Hunt said that among the other questions he would like to ask was whether Dr. Patrick had found any etiological factor in these cases which might reflexly have caused the spasm. When Brissaud separated the tic movements of the face from the spasms he spoke of the reflex origin of these spasms; the sensory limb of reflex arc being represented by the fifth nerve. Babinski, in a recent contribution to this subject, regards the facial spasm as due to some lesion, either of the nucleus or of the trunk of the facial nerve, regarding the trunk of the facial nerve as purely motor. Last year before this Association among other sensory functions of the seventh cranial nerve, Dr. Hunt spoke of reflex facial spasm, and he thinks that may have some practical importance in the treatment of these cases. In going over the literature of facial spasms he has been surprised at the number of cases in which there has been associated auditory disturbances, rupture of the tympanum, accumulation of cerumen in the external canal, chronic otitis media or nerve deafness. There is also another group of cases of central origin, notably two cases of aneurism with pressure on the facial nerve at the base of the skull, without any pressure on the pons or on the fifth nerve. Dr. Hunt could not understand how the irritation of the motor nerve trunk could induce a state of more or less rhythmical and recurrent spasms in the facial nerve. He believes that the sensory mechanism of the facial nerve plays the afferent factor in the production of some types of facial spasm, so that we must not only consider the fifth nerve as an afferent factor, but also the seventh nerve. This sensory mechanism may also help us to a proper understanding of the secondary contractures and twitchings of facial palsy. The sensory mechanism of the seventh cranial nerve consists of the geniculate ganglion, the nerve of Wrisberg, the petrosal branches to the middle ear and sensory fibers passing out into the motor trunk, and distributed to the region of the concha, auditory canal and tympanic membrane.

Hence the importance of the internal, middle and external auditory mechanism as a reflex factor in facial spasms.

Dr. W. G. Spiller remarked that we should not confound facial spasm such as Dr. Patrick had described with the tic occurring after a facial palsy. The latter was never observed unless some return of voluntary power was present but was incomplete. Lipschitz has shown that the tic of the lower part of the face developing after facial palsy is synchronous with winking. When regeneration occurs it is not probable that the same association of nerve fibers and nerve cells in the facial supply exists that was present before the palsy. Nerve cells formerly in connection with fibers of the upper branch of the facial nerve may in the process of regeneration be brought into association with fibers of the lower branch. Dr. Spiller had observed repeatedly that the tic of the lower part of the face in old facial palsy occurs at the same time as winking.

Dr. Putnam said he would like to express his acquiescence in what Dr. Spiller stated and would suggest that strychnia might affect the sensory or motor irritation. Strychnia causes increased motion not by acting on the motor, but the sensory centers.

Dr. Patrick, in closing, stated that he does not know how the alcohol acts. He has an idea that it blocks off something after the injections, just as stretching the nerves in facial neuralgia blocks off something. It is a pathological filter which holds back what we don't want and lets what we do want get through. Dr. Patrick said he does not know anyone who has thoroughly worked out the action of alcohol on the nerve fibers. In years gone by he tried stretching, with the same result as Dr. Putnam. The spasm returned with voluntary motion. He does not put forward the alcohol treatment as a radical cure. If the patients remain cured for a long time he does not see any objection to repeating the injection if necessary. He has not made the injection for Bell's palsy and would not. He had injected 15 or 20 minims of a 75 per cent. solution in the first case; this he thinks is too strong. Afterwards he used 40 to 50 per cent. He uses 15 to 20 minims because he puts in the needle and pulls it out a little way and then puts it in again, and so distributes the amount. During the several punctures he did not get any distinct twitching in the facial muscles. He presumes if the hypodermic needle had hit the nerve it would have caused sufficient movement to be perceptible to the operator. As to the etiology, he has nothing to say. The injections are easily made and the results to date are so gratifying that Dr. Patrick resolved to report the cases. He disclaimed any originality in the idea of alcohol injections in the treatment of facial spasm, stating that the idea was not his.

(To be continued)